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Title of Thesis: "Anxiety Sensitivity, Body Vigilance, Interoceptive Acuity, and Cardiovascular Reactivity in the Genesis of Panic"

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ABSTRACT

Title of Thesis: Anxiety Sensitivity, Body Vigilance, Interoceptive Acuity, and Cardiovascular Reactivity in the Genesis of Panic

Julie M. Storey, Master of Science, 2000

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Cognitive conceptualizations of panic require both the experience of arousal symptoms and their catastrophic interpretation. The tendency to interpret arousal symptoms as threatening is known as anxiety sensitivity (AS), but it is unclear if increased vigilance, greater physiological reactivity, or enhanced perception are responsible for reported physiological symptoms. Each of these mechanisms has been empirically supported in clinical, but not nonclinical, populations. The current investigation examined the ability of AS, body vigilance, cardiovascular reactivity, and interoceptive acuity to predict fearful responding to a 35% CO_2 inhalation in a nonclinical population. A main effect was found for AS ($R^2 = .13$; $p < .01$). Two interaction effects were found (AS x Heart Rate, $\Delta R^2 = .05$, $p < .05$; AS x Diastolic Blood Pressure, $\Delta R^2 = .05$, $p < .05$). Results support cognitive theories of panic and suggest physiological reactivity combined with AS elicit more fearful responses than either alone.

ANXIETY SENSITIVITY, BODY VIGILANCE,

INTEROCEPTIVE ACUITY, AND CARDIOVASCULAR

REACTIVITY IN THE GENESIS OF PANIC

by

Julie M. Storey, Capt, USAF

Thesis submitted to the Faculty of the
Department of Medical and Clinical Psychology Graduate Program of the
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Introduction

The central features of a panic attack, as defined by the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV; American Psychiatric Association, 1994), are intense fear accompanied by somatic and/or cognitive symptoms. This theoretically singular entity is estimated to affect 28-34% of individuals across their lifetimes (Norton, Harrison, Hauch, and Rhodes, 1985) and is linked to a broad range of psychological and physical outcomes. While some individuals report an isolated, uncomfortable event, others report a debilitating disorder (e.g., panic disorder).

Since the inclusion of panic attacks and panic disorder in the DSM-III (Diagnostic and Statistical Manual of Mental Disorders, Third Edition; American Psychiatric Association, 1980), this amalgam of physical and emotional complaints has been a topic of much research and debate. Significant advances have been made in our understanding of the maintenance and clinical course of these entities (McNally, 1994). Cognitive theory, applied to panic attacks and panic disorder in the mid-1980's, proved to be an important impetus for panic research. Cognitive conceptualizations of panic suggest that a panic attack results when benign physical symptoms are catastrophically interpreted (Clark, 1986; Reiss and McNally, 1985). Two key components identified by this conceptualization are: the *experience* of physical symptoms, and the *interpretation* of them in a catastrophic framework.

Physical Symptoms in Panic

Much debate abounds regarding the actual and perceived physical symptoms "experienced" by individuals plagued by panic attacks. Ehlers (1993) identified three hypotheses regarding increased symptom perception in panic disorder. Patients with

panic disorder may be more apt to perceive autonomic sensations because of: increased attention to levels of physiological arousal; enhanced ability to perceive normal physiological aberrations; and/or greater physiological reactivity to situations experienced as anxiogenic.

Body vigilance. In cognitive theory, a systematic preference for certain types of information is known as an *attentional bias* (Clark and Fairburn, 1997). An attentional bias for somatic changes has been suggested and observed to play a role in the perception of physical sensations (Ehlers, 1993). Pennebaker, Gonder-Frederick, Cox, and Hoover (1985) observed that increased attentional focus on the body increases the likelihood of perceiving potentially threatening interoceptive cues. Although this assertion has historically been reserved for patients with hypochondriacal concerns (i.e., concerned with the onset or presence of serious physical illness), recent evidence suggests it may be true for panic disorder patients as well as individuals with subclinical somatic concerns.

Investigations into panic disorder have labeled this tendency to selectively attend to somatic changes as *body vigilance* (Schmidt, Lerew, and Trakowski, 1997). In a multi-study investigation of the relationship between body vigilance and anxiety pathology, Schmidt and colleagues (1997) found body vigilance to be normally distributed in a nonclinical sample and to be related to a history of spontaneous panic attacks, anxiety sensitivity, and anxiety symptomatology. Additionally, individuals with panic disorder reported higher levels of body vigilance relative to social phobics and nonclinical controls and showed reductions in body vigilance associated with reported reductions in panic symptoms following a cognitive-behavioral intervention.

It has additionally been suggested that this heightened vigilance exists only for somatic disturbances related to autonomic nervous system arousal (e.g., heart palpitations and shortness of breath) but not for non-autonomic sensations (e.g., muscle aches or stomach pains) in panic disorder patients (Pilkington, Antony, and Swinson, 1998). In a study involving anxiety disordered patients and nonclinical volunteers, Pilkington and colleagues found vigilance for autonomic sensations to be significantly elevated in panic disorder patients relative to nonclinical controls, specific phobic patients, social phobic patients, and obsessive-compulsive patients. Panic disorder patients were no more vigilant for non-autonomic bodily sensations than nonclincal controls.

These data suggest an attentional bias for symptoms of autonomic arousal in panic disordered patients. Evidence for such a bias, and indirect evidence for the relevance of body vigilance in the maintenance of panic disorder, can be gleaned from Horenstein and Segui (1997) and Asmundson, Sandler, Wilson, and Walker (1992). Both of these studies were reaction time studies using panic disorder patients, clinical controls, and nonclinical controls. Two words were presented simultaneously to the subject, one word was a neutral word, and the other a threatening word. A probe dot then replaced one of the words, and the latency to detection of the probe was measured. Panic disorder patients were quicker to detect the probe when it replaced the threatening word rather than the neutral word. Asmundson et al. (1992) found a similar pattern, but only for physically threatening words (e.g., collapse and death). Socially threatening words (e.g., failure and stupid) did not enhance reaction time.

Although a relatively young construct, these preliminary investigations suggest that body vigilance to be a behavioral sequela of catastrophic cognitions and a mechanism by

which individuals with panic disorder detect changes in their somatic states leading to self-perpetuating anxiety responses.

Interoceptive acuity. Whereas body vigilance involves *attention* to somatic perturbations, interoceptive acuity involves *accuracy* in detecting such changes. Although results have been somewhat mixed, some research suggests that panic disorder patients, in addition to heightened vigilance, may also have an enhanced accuracy in the perception of internal somatic cues (Ehlers and Breuer, 1992). Although empirical support is relatively recent, the notion that certain people are more attuned to changes in their soma is not a new one. Tyrer (1976) reports that patients with somatic anxiety are better at perceiving bodily states than other subjects, and Shands and Schor (1982) described panic disorder patients as "interoceptive experts, being able to describe changes in almost every organ system and region of the body" (p. 108). Because cardiovascular symptoms are common during panic, evaluation of interoceptive acuity in panic disorder patients has typically focused on the accuracy of detecting one's heart rate (Ehlers and Breuer, 1992; McLeod and Hoehn-Saric, 1993).

Early studies of interoception began as studies of vigilance – focusing more on patient's self-reported awareness and not on their objective accuracy. These early investigations suggest that panic disorder patients *believe* they are more aware of somatic (usually cardiovascular) aberrations and act accordingly (fearfully) when they "perceive" such changes. For example, subjects with high state or trait anxiety have shown greater *reported* cardiac awareness than non-anxious subjects (Schandry, 1981; Montgomery and Jones, 1984), and nonclinical subjects who report good heartbeat perception are emotionally more responsive to stressors than subjects who do not (Katkin, 1985;

Schandry, 1983). Ehlers and Breuer (1992) found panic patients, as well as infrequent panickers, reported greater baseline cardiac sensitivity than either patients with other anxiety disorders or nonclinical controls. Additionally, self-reported cardiac awareness has been found to be related to the degree of agoraphobic avoidance behavior among frequent and infrequent panickers. Anxiety patients, but not healthy volunteers, report increases in anxiety and exhibit increases in physiological arousal when they hear false auditory feedback indicating a surge in heart rate (Ehlers, Margraf, Roth, Taylor, and Birbaumer, 1988). Although such self-reported awareness indirectly suggests heightened accuracy, self-reports of interoceptive sensitivity and objective measures of accuracy tend to have low correlations (Shands and Schor, 1982; Tyrer, 1973, 1976; Whitehead, Drescher, Heiman, and Blackwell, 1977).

Some studies investigating objective accuracy suggest panic disorder patients are more accurate at perceiving their heart beats than patients with simple phobias, infrequent panickers, and nonclinical controls, although panic disorder patients are not typically completely accurate (Ehlers and Breuer, 1992). Ehlers (1995), in a prospective evaluation of panic disorder patients, infrequent panickers, simple phobics, remitted panickers, and controls found maintenance of the disorder as well as relapse to be associated with good heartbeat perception, in addition to anxiety sensitivity, and degree of avoidance.

Although evidence suggests panic disorder patients have heightened awareness of their cardiac sensations, it is unclear whether increased cardiac awareness is present before the onset of panic attacks or whether it is acquired later in the course of the disorder (Ehlers and Breuer, 1992). It has been hypothesized that interoceptive acuity

may evolve from frequent pulse taking or changes in attentional focus that increase the probability that panic patients will be aware of normal cardiac changes (e.g., tachycardias, arrhythmias) that occur frequently in many people but go unnoticed (Ehlers and Breuer, 1992). As interoceptive acuity improves, such changes are more likely to be detected by vulnerable subjects (i.e., subjects with high anxiety sensitivity), and a panic attack is apt to be triggered. At least one study (Ehlers and Breuer, 1992) found infrequent panickers to be no better than nonclinical controls in detecting their heart rate, suggesting accuracy of heart rate detection, if it in fact exists in panickers, may be a result of long-term experience with panic attacks more than a cause.

Cardiovascular reactivity. Changes from baseline functioning of the heart or vasculature induced by experimental or in vivo demands are known as *cardiovascular reactivity*. Largely due to the fact that symptoms frequently reported by anxiety disorder patients suggest an increase in cardiovascular functioning (e.g., palpitations, chest pain, chest tightness), the literature is replete with investigations into the relationship between cardiovascular measures and anxiety symptomatology. Although results are not entirely consistent, a large portion of the evidence suggests baseline cardiovascular profiles, which include heart rate and blood pressure measures, of individuals with anxiety pathology do not differ significantly from those of nonclinical controls (Braune, Albus, Froehler, and Hoen et al., 1994; Hoehn-Saric and McLeod, 1988; Sandler, Wilson, Asmundson, Larsen, et al., 1992). However, it appears as individuals with anxiety pathology begin to experience anxiety, they may experience larger changes in their cardiovascular functions, including greater elevations in heart rate, systolic blood

pressure and diastolic blood pressure (Hoehn-Saric and McLeod; 1988; Pauli, Marquardt, Hartl, Nutzinger, Holzl, and Strian, 1991).

Heart rate reactivity. Several laboratory studies have demonstrated the presence of elevated heart rate responses to physiological challenges in panic disorder patients when compared to nonclinical controls. Patients with panic disorder have been shown to demonstrate greater heart rate reactivity to treadmill exercise (Taylor, King, Ehlers et al., 1987), to an orthostatic challenge (Stein, Papp, Klein, et al., 1992), to a biological challenge (vanZijderveld, TenVoorde, Veltman, and van Doornen, 1997), and to certain psychomotor tasks (Hoehn-Saric, McLeod, and Zimmerli, 1991) when compared with nonclinical controls, social phobics (Veltman, van Zijderveld, Tilder, and van Dyck, 1996), and blood phobics (Friedman, Thayer, Borkoved, Tyrrell, Johnson, Columbo, 1993; Friedman and Thayer, 1998). As with many constructs implicated in anxiety pathology, however, it remains unclear if this increased reactivity is a risk factor for panic or a consequence of cognitive (fear) and behavioral (avoidance of exercise) sequelae of panic.

Blood pressure reactivity. Studies of blood pressure reactivity, especially in relation to diastolic blood pressure reactivity, have yielded equivocal results. Although most investigations report elevated systolic blood pressure reactivity in panic disorder patients during ambulatory monitoring and in response to a variety of stressors (e.g., a mental stress task, epinephrine challenge; Bystritsky, Craske, Maidenberg, Vapnik, and Shapiro, 1995; Hoehn-Saric et al., 1991; Shear et al, 1992; White and Baker, 1987; vanZijderveld, TenVoorde, Veltman, and van Doornen, 1997), diastolic reactivity differs depending upon the type of stressor and the time of measurement (Bystritsky and Shapiro, 1992;

Yeregani, Meiri, Pohl, Balon, Desai, and Golec, 1990; vanZijderveld, TenVoorde, Veltman, and van Doornen, 1997). However, Bystritsky and Shapiro (1992) reported elevated diastolic blood pressure reactivity in panic disorder patients during a carbon dioxide challenge, and three different investigations of ambulatory blood pressure indicate significantly elevated diastolic blood pressure reactivity prior to and during panic in a naturalistic setting (Bystritsky et al, 1995; Shear et al, 1992; White and Baker, 1987).

Although evidence to date suggests cardiovascular reactivity's role in the maintenance of anxiety pathology, few studies have investigated its role in development of these disorders. Sandler, Wilson, Asmundson, Gordon, Larsen et al. (1992) compared infrequent panickers to nonpanicking controls and discovered that infrequent panickers did not evidence the heightened autonomic activation that is often found in individuals with panic disorder. This study, while not conclusive, suggests that cardiovascular reactivity may be a systemic response to repeated, intense autonomic arousal and not a causal mechanism in the development of panic. To date, no studies have prospectively investigated cardiovascular reactivity, and few have reported its relationship to fear in a nonclinical sample. Shostak and Peterson (1990) investigated the relationship between anxiety sensitivity and physiological changes in nonclinical subjects following a mental arithmetic task and found comparable muscle activity and systolic blood pressure across three levels of anxiety sensitivity. However, one might logically question the relevance of a mental stress task for individuals high in anxiety sensitivity. Similarly, Asmundson, Norton, Wilson, and Sandler (1994) failed to find heart-rate reactivity differences between high and low anxiety sensitive nonclinical subjects following a hyperventilation challenge. In this investigation, Asmundson and colleagues failed to provide evidence of

arousal in either group, bringing into question the efficacy of the stressor and thus the accuracy of their conclusions.

Catastrophic Interpretation of Symptoms in Panic

The second component of the cognitive conceptualization of panic is the misinterpretation of innocuous, internal bodily sensations as threatening (Barlow, 1988, Clark, 1986). Identified as threatening, these sensations may trigger anxiety, increased arousal, and self-perpetuating fear. According to this model of panic, the mere experience of physical arousal is insufficient to elicit panic. The individual must believe that the physical arousal may have negative consequences.

The extent to which an individual believes that autonomic arousal can result in catastrophic consequences in known as anxiety sensitivity (Reiss and McNally, 1985). Individuals high in anxiety sensitivity may believe that shortness of breath signals suffocation or that heart palpitations indicate a heart attack, whereas those low in anxiety sensitivity experience these sensations as uncomfortable but innocuous (McNally, 1994). Evidence suggests that anxiety sensitivity is a stable, trait-like characteristic that emerges from experiences (both personal and observational) that yoke aversive consequences with arousal (McNally, 1994).

Empirical support for anxiety sensitivity's role in panic is fairly strong. Elevated anxiety sensitivity has been shown to be typical of anxiety disorders in general (Taylor, Koch, and McNally, 1992), with panic disorder patients scoring two standard deviations greater than controls, and significantly higher than patients with generalized anxiety disorder (McNally, Amir, Louro, Lukach, Riemann, and Calamari, 1994). Additionally, anxiety sensitivity has been shown to predict diagnostic severity of panic disorder (Jones

and Barlow, 1991, c.f., Telch, Silverman, and Schmidt, 1996) and has been shown to diminish with the reduction of anxiety symptomatology (McNally and Lorenz, 1987; Telch, Lucas, Schmidt, Hanna, Jaimez and Lucas, 1993).

Empirical evidence garnered via multiple methods and across multiple populations suggests anxiety sensitivity may play a prominent role in the pathogenesis and the maintenance of panic disorder (McNally et al., 1998). Most notably, prospective investigations have identified anxiety sensitivity as a risk factor for the development of panic attacks and panic disorder (Donnell and McNally, 1990; Ehlers, 1995; Maller and Reiss, 1992; Schmidt et al., 1997). For example, Ehlers (1995) found high baseline anxiety sensitivity to be associated with the occurrence of a first panic attack during a one-year follow-up period. In the laboratory, anxiety sensitivity has been found to predict emotional and physical responding to various biological challenges regardless of panic history and diagnostic status (Donnell and McNally, 1989; Schmidt and Telch, 1994; Rapee and Medoro, 1994; Telch and Harrington, 1993; Telch et al., 1996; Holloway and McNally, 1987). Telch and Harrington (1992) found subjects with high anxiety sensitivity without a history of panic attacks exhibit rates of CO_2 -induced panic comparable to panic disorder patients. Eke and McNally (1996) found that psychological variables reflecting fears of bodily sensations are better predictors of response to carbon dioxide challenge than either behavioral sensitivity to carbon dioxide or general trait anxiety.

While research concerning anxiety sensitivity, body vigilance, interoceptive acuity, and cardiovascular reactivity has shed considerable light upon the maintenance of panic, the focus has only recently turned to the enlightenment of its pathogenesis (Maller and

Reiss, 1992; Schmidt, Lerew, and Jackson, 1997; Ehlers and Breuer, 1992; Schmidt et al., 1997). Additionally, prior investigations into risk factors for anxiety pathology have to date involved the sole consideration of the singular effects of each of the suspected risk factors. Very few studies have investigated the relationship between anxiety sensitivity, cardiac reactivity, and heartbeat perception in nonclinical subjects.

In one of the few existing investigations into these constructs in a nonclinical sample, Sturges and Goetsch (1996) exposed nonclinically anxious (i.e., high anxiety sensitivity) women and nonanxious (i.e., low anxiety sensitivity) women to caffeine-induced arousal and compared heart rate and skin conductance reactivity and interoceptive accuracy and failed to find an effect. However, methodological difficulties, such as the failure to measure blood pressure and the allowance of practice trials on the heart beat tracking task, may have masked or ignored important differences between the groups. The authors also did not investigate interactive effects between the variables of interest, and they failed to include a measure of symptoms and subjective distress in relation to the caffeine challenge.

Thus, the principle aim of the present study was to investigate the hypotheses proposed by Ehlers (1993) by evaluating the singular and interactive effects of anxiety sensitivity, body vigilance, heartbeat perception, and cardiovascular reactivity in predicting fearful responding to a biological challenge in individuals with no history of spontaneous panic or any anxiety disorder diagnosis.

Study Hypotheses

Ehlers's (1993) hypothesized that individuals with panic disorder are more likely to perceive physical symptoms for one of three reasons: they pay more attention to them;

are more accurate at perceiving them; or they are more reactivity physiologically to anxiogenic physical stimuli. Additionally, recent research into the role of cognitions in panic suggests the catastrophic interpretation of physical symptoms is important in the genesis of anxiety and fear. Therefore, it was hypothesized that:

- Catastrophic interpretation of symptoms will predict fearful responding to a carbon dioxide challenge in a nonclinical sample.
- Catastrophic interpretation will mediate the roles of somatic vigilance, interoceptive acuity, and cardiovascular reactivity in self-reported fear. That is, neither body vigilance, heartbeat perception, nor cardiovascular reactivity (i.e., changes in heart rate, systolic and diastolic blood pressure) will predict fearful responding unless they are catastrophically interpreted.

Methods

Sample and Procedures

72 (27 males and 45 females) non-clinical subjects from Washington, D.C. and Montgomery County, Maryland participated in the study. Potential subjects were contacted by phone from a list of 1,516 phone numbers selected randomly by city block and purchased from a national survey sampling company (See Table 1). Of the 1,516 phone numbers, 107 eligible residences expressing interest in the study were identified. These 107 households contained a total of 135 eligible people.

Respondents were administered a brief, preliminary telephone interview to determine their eligibility. The data presented in this paper were collected as part of a

Table 1

Contact Results for 1516 Random Phone Numbers

Status	Count
Business (nonresidential) Numbers	131
Couldn't Contact	639
Disconnected Numbers	288
Fax Numbers	87
Hang-ups	20
Ineligible Households Age: 156 Medical: 5 Psych Hx: 5 Other: 37	203
Not Interested	41
Interested/Eligible households	107
Total	1516

larger, prospective study of risk factors for anxiety pathology. Based upon epidemiological data that suggest young adults to be at highest risk for the development of anxiety pathology (Weissman and Merikangas, 1986; Burns and Thorpe, 1977), eligible respondents were between the ages of 18 and 35 and without a history of spontaneous panic or a DSM-IV Axis I disorder. Additionally, eligible respondents were without significant, current medical illness (e.g., renal, cardiovascular, neurological, or pulmonary disease), which could account for differences in physiological indices at baseline and in response to the biological challenge task. Due to the format of the current protocol, all respondents were able to comprehend spoken and written English. All eligible individuals living in the selected household were accepted into the study and were asked to come in for one lab session.

Physiological Measures

Vital capacity. A Respirodyne II Plus respirometer and disposable flow sensors were used to measure each subject's vital capacity (VC). VC is the maximum volume of air that can be moved in and out of the lungs and is measured in liters. VC was assessed three times and averaged to yield a VC index.

Heart rate and blood pressure. Heart rate, systolic and diastolic blood pressure were recorded using a Critikon Dynamap Vital Signs Monitor, Model 1846 SX. The monitor was set to read blood pressure and heart rate at one-minute intervals during the 10-minute baseline. Measures were suspended during the interoception protocol (see below). Following each inhalation, 2 immediate blood pressure and heart rate measures were taken.

Vagal tone. A single channel, three lead signal from an electrocardiographic pre-amplifier (VTM II, Scope Services, Bethesda, Maryland), provided visual, computerized numeric output of heart rate which allowed for precise assessment of the subject's interoceptive accuracy.

Interoceptive accuracy. Interoceptive accuracy was assessed via the mental tracking paradigm designed by Schandry (1981) and modified by Ehlers and Breuer (1992). Subjects were instructed to count their heartbeats silently during intervals of 35 seconds, 25 seconds, and 45 seconds without taking their pulses or using other perception strategies. In order to minimize random guessing, subjects were specifically instructed to count only the heartbeats they actually perceive and not to count just because they know their heart should be beating (Zoellner and Craske, 1999). Each interval began with the experimenter asking if the subject was ready, and upon a positive response stating, "Go."

Simultaneously, another experimenter counted actual R-waves from electrocardiogram (ECG) recordings of the VTM-II. Interoceptive accuracy was defined as the absolute value of the actual number of heartbeats minus the number of perceived heartbeats divided by the actual number of heartbeats multiplied by 100. The resulting quotient is identified as heart beat accuracy (HBA).

Psychological Measures

Structured clinical interview. In order to assess Axis I psychiatric disorders, the Structured Clinical Interview for Axis I DSM-IV Disorders, Patient Edition (SCID-I/P-Version 2.0; First, Spitzer, Gibbon, and Williams, 1996) was administered to all subjects by a graduate student trained in its administration. The SCID-I/P has shown high validity and good test-retest reliability in diagnosing Axis I disorders in a variety of populations (Williams, Spitzer, and Gibbon, 1992).

Acute Panic Inventory. The Acute Panic Inventory (API) is a 24-item inventory for assessing symptoms of arousal associated with panic attacks (Liebowitz et al., 1984). Subjects rate the severity of each symptom from 0 (absent) to 3 (severe). Examples include, "Did you feel faint?", and "Were you afraid of dying?". The API includes two self-report visual analog scales of the subject's current level of fear and highest level of fear (0 – no fear at all, 100 – extreme fear).

Anxiety Sensitivity Index. The Anxiety Sensitivity Index (ASI) is a 16-item questionnaire that measures fear of somatic symptoms related to arousal (Peterson and Reiss, 1992). Each item assesses a concern about the possible negative consequences of anxiety symptoms on a 0 to 4 point likert scale. This test is scored by summing each response to provide a total score. The ASI has demonstrated adequate internal

consistency (Telch, Shermis, and Lucas, 1989) and test-retest reliability (Maller and Reiss, 1992). The ASI appears to tap into fear of anxiety symptoms as opposed to state or trait anxiety (see McNally, 1994). The mean for nonclinical samples has been observed to be 19 (Peterson and Reiss, 1992).

Body Vigilance Scale. The Body Vigilance Scale (BVS; Schmidt et al., 1997) is a 4-item self-report inventory that was designed to assess attentional focus to internal body sensations. The first three items assess degree of attentional focus, perceived sensitivity to changes in bodily sensations, and the average amount of time spent attending to bodily sensations. The fourth item involves separate ratings for attention to 15 sensations (e.g., heart palpitations) that include all of the physical symptoms described for panic attacks in the DSM-IV (American Psychiatric Association, 1994). Scores on item 3 are divided by 10. Ratings for the 15 sensations are averaged to yield a single score for item 4. The BVS total score is the sum of items 1-4. The BVS has been shown to have adequate test-retest reliability and high internal consistency, and the mean score for nonclinical subjects has been reported to be 18.0 (Schmidt et. al., 1997).

Biological Challenge Procedure

Prior to exposing the subject to the CO_2 challenge, the subject's vital capacity (VC) was measured. The experimenter provided instructions and a demonstration of the VC procedure. The subject inhaled as much air as possible, placed his/her mouth around the flow sensor making a tight seal, and exhaled through the flow sensor. Following three VC measurements, the subjects were given the following instructions: "Next you will take two deep breaths. One breath will be of normal room air, and the other will be a mixture of oxygen and carbon dioxide. The oxygen and carbon dioxide mixture will

produce some short-lived sensations. You will not be told which one you will receive first in order to ensure your response will not be biased. As I said before, you will take one single breath of each gas. I will ask you to hold the inhalation while I count to five, and then you can exhale." Next, the experimenter demonstrated taking a vital capacity breath from the venti-comp bag. The order of presentation of the two gases was determined by coin flip prior to the initiation of the protocol. The experimenter then assisted the subject in taking a vital capacity breath of each of the gases (35% carbon dioxide and 65% oxygen, and room air). The mixture was delivered to subjects via 4.8 liter venti-comp bags filled to capacity. The patient, with nostrils closed, exhaled all of the air in his/her lungs and then inhaled from the venti-comp bag via a one-way flow valve with the goal of inhaling as much of the mixture as possible. The challenge phase consisted of the inhalation period plus 30 seconds following CO_2 inhalation. The CO_2 intake volume was assessed by measuring the amount of CO_2 remaining in the venti-comp bag. The API was obtained after each phase. The patient was then disconnected from the apparatus.

Protocol Overview

After reviewing the consent forms and administering the SCID-I/P, subjects were given a battery of self-report inventories including the ASI, BVS, and the STAI. After completing these questionnaires, subjects were then connected to the VTM-II and dynamap and asked to sit still and quietly for a 10-minute baseline. At the end of the baseline period, subjects completed the Interoceptive Accuracy paradigm and then the CO_2 challenge.

Data Analytic Strategy

Total scores were calculated for the ASI and the BVS according to their standardized scoring algorithms. Measures of reactivity for each of the cardiovascular indices (heart rate, diastolic blood pressure, and systolic blood pressure) were calculated by partialling out the variance in scores accounted for by baseline measures (i.e., for each cardiovascular measure, a linear regression analysis was performed using the measure's baseline level and room air reactivity level as independent variables and the CO_2 reactivity level as the dependent level. The unstandardized residuals of these regressions were used as the reactivity variable for each of cardiovascular indices).

Two hierarchical multiple regression analyses were performed to examine the predictors of self-reported fear in response to the CO_2 challenge. The dependent variable was the subject's level of fear reported immediately after the biological challenge on the visual analog scale of the API. The six independent variables for this analysis represented fear of arousal symptoms (ASI) and Ehlers's (1993) hypothesized mechanisms for perception of physical symptoms (BVS, HBA, ΔHR, ΔDBP, ΔSBP). The independent variables were entered stepwise, and the order of entry was based on cognitive theory and prior research findings discussed in the Introduction.

The second regression analysis was designed to identify interaction effects between the ASI and each of the other five independent variables on the dependent variable, post-CO_2 self-reported level of fear. The independent variables were entered in six steps. The first step contained the main effects for ASI, BVS, HBA, ΔHR, ΔDBP, ΔSBP. Steps two through six contained the cross-products of the ASI and each of the

other five variables. The cross products were entered stepwise, and the order of entry was based on cognitive theory and prior research findings discussed in the Introduction.

Results

Descriptive Statistics

Table 2 gives descriptive statistics for the initial sample of 72 subjects. The sample primarily included young, healthy Caucasian women with at least a high school diploma. The sample means on the anxiety sensitivity index (ASI), the body vigilance scale (BVS), and baseline cardiovascular measures were within the normal range but were lower than previously reported means for nonclinical subjects (Table 2).

Table 2

Sample Descriptive Statistics

	n	%
Gender		
Female	45	62.5%
Male	27	37.5%
Ethnicity		
Caucasian	40	54.9%
African American	20	28.1%
Hispanic	5	7.2%
Asian	3	4.2%
Other	4	5.6%
	Range	M(SD)
Age (yrs)	18-35	26.9(4.6)
Education (yrs)	11-18	14.7(2.1)

Missing Data

As Table 3 indicates, each predictor variable had missing data points. Most of these missing data points were due to time constraints and equipment difficulties. None of the missing data points were due to subject refusal to complete the protocol, and no single subject was missing more than 2 data points. Pairwise deletion of subjects with missing data yielded a reduced sample of 48. As a result, statistical analyses included replacement of missing data with the sample mean for that measure.

Table 3

Descriptive Statistics for the Predictor Measures

			Missing data	
Variable	Range	M(SD)	n	%
Anxiety Sensitivity Index	1 – 37	14.58(8.2)	7	9.7
Body Vigilance Scale	0 - 35.3	10.81(8.2)	5	6.9
Heartbeat Accuracy (% inaccurate)	3.4 - 100	87.12(21.0)	4	5.5
Baseline Systolic Blood Pressure	84 - 150	111.53(13.1)	7	9.7
Post CO_2 Systolic Blood Pressure	91 - 164	120.48(16.4)	8	11.1
Baseline Diastolic Blood Pressure	48 – 92	66.91(9.1)	7	9.7
Post CO_2 Diastolic Blood Pressure	52 – 99	73.46(10.0)	8	11.1
Baseline Heart Rate	41 – 96	64.76(11.0)	7	9.7
Post CO_2 Heart Rate	45 - 104	67.77(11.4)	6	8.3

Correlation Matrix

Table 4 contains the correlations among the six independent variables. Significant correlations ($p<.05$) existed between the ASI and SBP reactivity, and SBP reactivity and HR reactivity.

Table 4

Correlation Matrix for Predictor Variables

Variable	1	2	3	4	5	6
1. ASI	--	.101	.077	-.099	-.270	.327*
2. BVS		--	.230	.035	-.035	-.105
3. HBA			--	-.163	-.172	.082
4. ΔHR				--	.152	-.365*
5. ΔDBP					--	.056
6. ΔSBP						--

*$p < .05$

Table 5

Summary of Hierarchical Regression Analysis for Variables Predicting Post CO_2 Fear ($N = 72$)

Variable	B	SE B	β
Anxiety Sensitivity Index (ASI)	.07	.02	.36**
Body Vigilance Scale (BVS)	-.03	.02	-.15
Heart Beat Accuracy (HBA)	.00	.01	-.06
Heart Rate Reactivity (ΔHR)	.03	.02	.18
Diastolic Blood Pressure Reactivity (ΔDBP)	.02	.02	.14
Systolic Blood Pressure Reactivity (ΔSBP)	.01	.02	.06

Note. $R^2 = .13$ for Step 1 ($p < .01$)

**$p < .01$

Main Effects

The anxiety sensitivity index (ASI), body vigilance scale (BVS), heartbeat accuracy index (HBA), diastolic blood pressure reactivity (ΔDBP), systolic blood pressure reactivity (ΔSBP), heart rate reactivity (ΔHR) were entered hierarchically in the order

indicated in Table 5. The ASI was the only significant predictor, accounting for 13.2% of the variance in self-reported fear after the CO_2 challenge. Adding each of the other predictors did not significantly increase the variance explained.

Table 6

Summary of Hierarchical Regression Analysis for Cross Products Predicting Post CO_2 Fear ($N = 72$)

Variable	B	SE B	β
Step 1			
Anxiety Sensitivity Index (ASI)	.079	.774	.44**
Body Vigilance Scale (BVS)	-.031	.022	-.17
Heart Beat Accuracy (HBA)	.000	.021	.00
Heart Rate Reactivity (ΔHR)	.033	.008	.19
Diastolic Blood Pressure Reactivity (ΔDBP)	.021	.021	.13
Systolic Blood Pressure Reactivity (ΔSBP)	.009	.020	.06
Step 2			
ASIxBVS	.000	.002	.02
Step 3			
ASIxHBA	.000	.001	-.23
Step 4			
ASIxΔHR	.007	.003	.58*
Step 5			
ASIxΔDBP	.005	.003	.54*
Step 6			
ASIxΔSBP	.004	.002	.42

Note. $R^2 = .21$ for Step 1; $\Delta R^2 = .05$ for Step 4; $\Delta R^2 = .05$ for Step 5 ($ps < .05$)

*$p < .05$. **$p < .01$.

Interaction Effects

Table 6 represents the second multiple regression analysis examining the effects of

the cross products of the ASI and the other five independent variables. ASIxΔHR cross product (Step 4) increased the R^2 value to .266, a significant improvement over Step 3, indicating anxiety sensitivity's effect on fear is not the same for different levels of heart rate reactivity. A graphical representation of the ASI/ΔHR interaction is depicted in Figure 1.

Similarly, the ASIxΔDBP cross product significantly increased the R^2 value to .312. A graphical representation of the ASIxΔDBP interaction is depicted in Figure 2. The addition of the other cross products did not contribute significantly to the model. The overall model explained 34.5% of the variance in post-CO_2 self-reported fear.

Discussion

Cognitive notions of panic assert the preeminence of catastrophic thought processes in the development and maintenance of panic, and the findings from the present study lend support to this notion. When considering anxiety sensitivity, cardiovascular reactivity, body vigilance, and interoceptive accuracy, anxiety sensitivity emerged as the only significant factor associated with self-reported fear of carbon dioxide-induced arousal. These results suggest anxiety sensitivity to be a dispositional factor that contributes to fearful responding to symptoms of arousal independent of panic history. Additionally, the findings are consistent with the formulation that when one perceives arousal as threatening, one is more apt to respond fearfully to arousal-inducing agents and activities regardless of one's prior history of panic attacks. These findings have been reported in previous studies investigating the role of anxiety sensitivity in nonclinical

subjects exposed to biological challenges (Holloway and McNally, 1987; Schmidt and Telch, 1994; Telch et al., 1996).

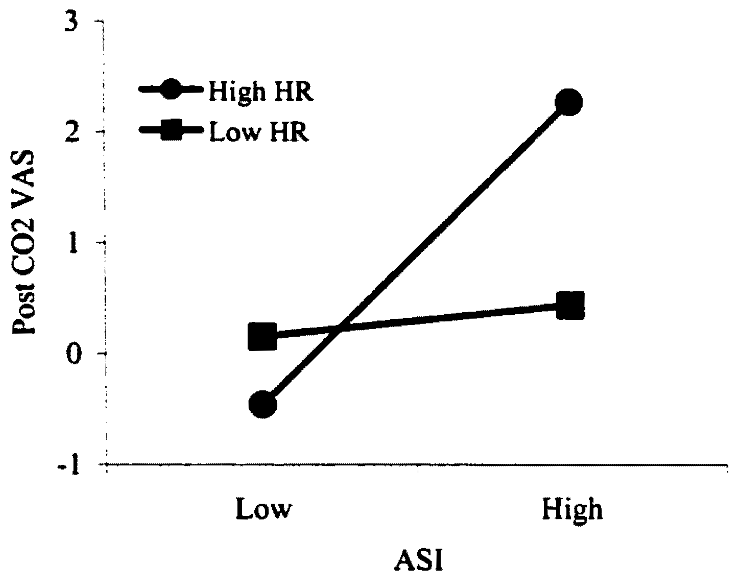

Figure 1. Interaction between anxiety sensitivity and heart rate reactivity

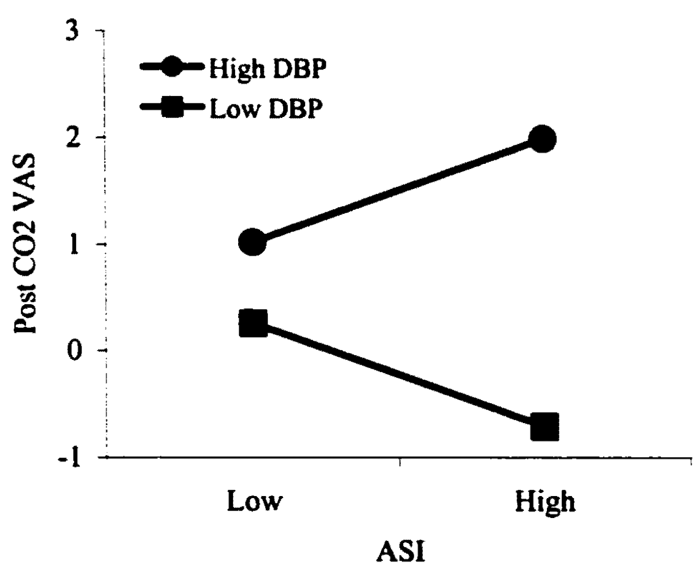

Figure 2. Interaction between anxiety sensitivity and diastolic blood pressure reactivity

The remaining results will be discussed in light of Ehlers's (1993) hypotheses regarding the role of heightened somatic perception in panic, namely increased vigilance, enhanced interoceptive acuity, and greater physiological reactivity. In the current, nonclinical sample, increased vigilance did not emerge as a significant factor associated with self-reported fear, neither singularly nor in interaction with anxiety sensitivity. The failure of vigilance to be associated with fearful responding to autonomic arousal suggests that vigilance, although potentially important in the maintenance of panic, is unlikely to be a substantial contributor to the development of panic.

The results suggest a similar conclusion with regard to interoceptive acuity. Although accuracy in detecting somatic changes may foster and sustain panicogenic cognitions and behaviors, the current findings do not implicate it as a significant contributor to fearful responding in subjects without histories of panic.

As for Ehlers's third hypothesis, that individuals who experience panic are more physiologically reactive, the current findings are more encouraging. Although none of the cardiovascular reactivity measures (i.e., heart rate, diastolic blood pressure, systolic blood pressure) singularly contributed to self-reported fear, heart rate reactivity and diastolic blood pressure reactivity, when considered with anxiety sensitivity status, appears to be a significant factor. As hypothesized, an anxiety sensitivity and diastolic blood pressure reactivity interaction emerged as a significant contributor to fearful responding to the carbon dioxide challenge, indicating high heart rate and diastolic blood pressure reactivity may increase fearful responding in individuals who also have high anxiety sensitivity. Because these findings emerged in this nonclinical sample, the results suggest these factors to be potential risks for the development of panic. This finding is

consistent with previous reports from ambulatory monitoring of individuals with panic disorder showing increased diastolic blood pressure reactivity during and near panic (Shear et al., 1992; White and Baker, 1987; Bystritsky et al., 1995) and with reports of elevated diastolic blood pressure in individuals with panic disorder in response to CO_2 challenge.

Additionally, a trend towards an anxiety sensitivity and systolic blood pressure interaction emerged in the analyses ($\Delta R^2 = .033$, $p < .09$), indicating the potential role of systolic blood pressure reactivity or the potential role of a general measure of reactivity (e.g., mean arterial pressure) that should be investigated in future studies with greater statistical power.

Limitations of the Study

The primary limitations of this study were the small sample size, missing data, and the cross-sectional design. The small sample size was a direct result of the methodology used for recruitment of subjects. Although random digit dialing techniques theoretically render a more representative sample, people today are inundated with tele-marketers and phone solicitors, so much so that many households were unwilling to speak to us and unwilling to return our phone calls. This method of subject recruitment could have resulted in an additional bias in that, intuitively, it seems that more anxious people would be less willing to commit to driving across a large metropolitan area to participate in a research study.

To some extent, this bias is supported by the sample's low scores relative to other nonclinical samples on the anxiety sensitivity index (McNally, 1996), and the body vigilance scale (Schmidt et al., 1997). Additionally, the sample's high degree of

inaccuracy in detecting heart rates, and lower than average baseline blood pressures and heart rates (Guyton and Hall, 1996) are indicative of such a selection bias. Subjects' low level of fearful responding to the CO_2 challenge could have also been influenced by the selection bias and other methodological limitations. For example, Telch and colleagues (1996) found that the emotional responding of nonclinical subjects with high anxiety sensitivity is influenced by perceived control (i.e., their ability to regulate their CO_2 intake).

In our study, subjects were told they could leave the study at any time, and they would be paid for the portion of the protocol they completed. This could have resulted in diminished emotional responding in an already nonanxious sample. The low scores on the SUDS scale indicating fear of the CO_2 challenge are suggestive of this fact (M = 1.2, SD = 1.9, possible range, 0-10). The recruitment methodology also resulted in a small sample size, which significantly diminished the statistical power of the analyses.

Missing data points were another source of concern in the current study. Due to the small sample size and the wide distribution of missing values, it was not possible to discard subjects with missing data. No single subject was missing more than 2 independent variable measures, but 24 subjects were missing at least one independent variable. Substitution with the mean was implemented for these subjects.

Conclusions

Although many questions remain regarding the genesis of panic and other anxiety disorders, the results of this study, interpreted in light of results from other nonclinical examples, have potential theoretical ramifications for current theory. The present findings suggest improved accuracy for detecting one's heart rate and vigilance for

symptoms of arousal may be common sequelae, but appear to be neither necessary nor sufficient links in the anxiety pathology chain. Prospective studies of nonclinical subjects will best be able to pursue these findings further. This study additionally provides added support to the notion that aberrant, catastrophic cognitions regarding the meaning of somatic symptoms may be necessary, and at times sufficient, to induce fearful responding to such sensations. As well, an individual's physiology, specifically one's cardiovascular reactivity, may be an important mediator in the genesis of fear.

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